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Postpartum pheochromocytoma crisis associated with cardiogenic shock and Sheehan's syndrome

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SUMMARY

We report a case of a woman in her mid-30s who developed severe cardiac shock hours after giving birth to her second child with the need for extracorporeal haemodynamic support. Initially, postpartum cardiomyopathy was suspected, and high-urgency heart transplantation was considered. However, the endocrine work-up and imaging revealed pheochromocytoma as the cause for acute heart failure that was completely reversible. Notably, the patient also developed Sheehan's syndrome with pituitary necrosis and sustained hypopituitarism, most likely as a consequence of the haemodynamic failure during pheochromocytoma crisis. While pheochromocytoma crisis is already an extremely rare peripartum complication, the current case is—to the best of our knowledge—the first report of pheochromocytoma associated with Sheehan's syndrome. This case also highlights the clinical conundrum that pheochromocytomas can be easily overlooked in pregnancy due to non-specific symptoms and confusion with pregnancy-related hypertension or hypertension-associated other diseases. Appropriate case detection is important, especially in pregnant women with early onset of hypertension.

BACKGROUND

Peripartum cardiomyopathy is a very rare condition with an incidence of 1 per 3000 to 1 per 15 000 live births and predominantly occurs during the last month of pregnancy or up to 5 months after delivery.[1 2] Previously identified risk factors are black race, multiparity, maternal age>30 years, twin pregnancies and history of hypertension, pre-eclampsia and eclampsia.[1 3] The prognosis depends on the ability of the left ventricular contractility to recover within the first months after the initial onset. The mortality rate is very high, with up to 50% and heart transplantation remains the last therapeutic option.[2] Postpartum cardiomyopathy is characterised by dilation of the left ventricle with global ventricular dysfunction and severely reduced ejection fraction (EF) resulting in the typical clinical manifestations of acute heart failure.[4 5] It can be challenging for clinicians to differentiate between peripartum causes (pre-eclampsia, gestational hypertension or peripartum cardiomyopathy) and other causes of cardiac insufficiency or pulmonary oedema (stress-induced or Takotsubo cardiomyopathy, pre-existing cardiac disease including mitral stenosis, pulmonary or amniotic fluid embolism or even pheochromocytomas).[4 6]

Phaeochromocytoma in pregnancy is even less frequent than peripartum cardiomyopathy, with an incidence estimated at 1 per 15 000 to 1 per 54 000 pregnancies.[7] However, maternal and fetal complications are high, with mortality rates up to 40–50% for both mother and fetus if undiagnosed or left untreated.[8]

This endocrine tumour is accompanied by a variety of symptoms including hypertension, headache, palpitations, excessive sweating, muscle tremor, vomiting, panic attacks, vasomotor disturbances and blurred vision.[8 9] Hypertension has been reported as the most common clinical manifestation of pheochromocytoma during pregnancy (88%); however, one-third of the affected women already had antepartum hypertensive crises.[10]

Another rare postpartum condition is Sheehan's syndrome, which presents as hypopituitarism due to pituitary necrosis as a consequence of ischaemia or haemorrhage during or after delivery.[11]

First described in 1937 by Harold L Sheehan, the condition remains often undiagnosed even for years mainly due to non-specific signs and symptoms such as lethargy, amenorrhoea or lactation problems. Every pituitary axis can be affected, but most common are lactotropic and somatotropic insufficiency, the latter presumably due to the peripheral location of somatotropic cells that are most vulnerable to ischaemic necrosis.[12]

Hypopituitarism is often irreversible, and deterioration of certain pituitary functions can also occur years after pregnancy.[13]

Notably, diabetes insipidus is rarely associated with Sheehan's syndrome. Therapy involves hormone replacement of the affected pituitary axis.[11 12]

To the best of our knowledge, the coincidence of both of these rare endocrine conditions, pheochromocytoma and Sheehan's syndrome, has never been reported before.

CASE PRESENTATION

A pregnant woman in her mid-30s at gestational week 41 was admitted to a community hospital in Austria and gave birth to her second child following an uncomplicated full-term pregnancy. Apart from recurring panic attacks and associated palpitations for about 4 years, her previous medical history was normal. Six hours after delivery, the patient suffered from nausea, tachycardia, and shortness of breath with a drop in peripheral oxygen saturation (85%) and the necessity of oxygen inflation. She was then transferred to the local intensive care unit (ICU). CT

showed no sign of pulmonary embolism but severe pulmonary congestion and a non-specific 9×9.5 cm septated cystic mass in the upper right abdomen, which was interpreted as haemorrhagic kidney cyst. Despite diuretic treatment and non-invasive ventilatory support, the respiratory situation deteriorated in the following hours, and the patient had to be intubated. Shortly after the intubation, she developed cardiogenic shock requiring mechanical cardiopulmonary resuscitation (CPR). Following the return of spontaneous circulation, the bedside echocardiography showed severely reduced left ventricular function with a calculated EF of less than 20%. Based on these results and the clinical presentation, peripartum cardiomyopathy was suspected, and the patient's circulation was supported by venoarterial extracorporeal membrane oxygenation (ECMO: cannulation sites: right femoral vein + right subclavian artery, with ECMO support of 3.5 L/min; 3000 rpm: fractional inspired oxygen: 60%; gas flow: 2 L/min).

Given that the cardiac function did not improve in the following days, our institution was contacted for high-urgency heart transplantation. To assess the neurological status, the patient was weaned from sedation and mechanical ventilation and showed no signs of neurological deficits after extubation. Hence, she was transferred to our ICU at the Division of Cardiothoracic and Vascular Anesthesia for heart transplantation evaluation. On arrival, the patient was dependent on full ECMO support but was awake and breathing spontaneously. Echocardiography revealed a global hypokinetic to akinetic left ventricle with severely reduced left ventricular function but normal right ventricular function, normal valves and a pericardial effusion of 1 cm. N-Terminal Pro-B-Type Natriuretic Peptide (NT-proBNP) was markedly elevated at 1852.6 pmol/L (0–14.7 pmol/L).

Given the differential diagnosis of Takotsubo cardiomyopathy or any other unknown—potentially reversible—acute cardiomyopathy, the patient was not immediately listed for heart transplantation. Initially, she had recurring haemodynamic fluctuations with hypertensive and severe hypotensive episodes requiring intermittent administration of vasodilators and vasopressors, respectively. However, in the following days after admission to our ICU, echocardiographic exams showed a continuous improvement of the left ventricular function, which allowed a subsequent weaning from the extracorporeal circulatory support. Nonetheless, a CT was performed to exclude an aortic injury given the volatile haemodynamic situation. The CT scan showed no aortic dissection but a large hypodense cystic mass (9×9.5×10.5 cm) in the area of the right kidney, which had been suspected as a haemorrhagic kidney cyst in the previous external CT scan.

As incidental findings, pulmonary embolism of the right upper pulmonary artery and a large thrombus in both iliac veins were now described, most likely from thrombotic streaks in the ECMO cannulas. The patient had no typical symptoms of pulmonary embolism but was subsequently anticoagulated.

Due to the reported mass in the right upper abdomen, the patient's history of panic attacks as well as palpitations and in light of the recent haemodynamic events, an endocrine work-up was performed and markedly elevated metanephrine (MN) and normetanephrine (NMN) levels were found in plasma highly suspicious of pheochromocytoma (table 1).

To confirm this diagnosis, MRI and an 18-fluorodopa positron emission tomography CT scan (F-DOPA PET/CT) were performed that revealed a 9×9.5×10 cm^3 cystic and haemorrhagic lesion in the right adrenal loge with distinct F-DOPA uptake, in accordance with pheochromocytoma of the right adrenal gland (figure 1). The radiographic imaging could be safely performed because the mother was not breastfeeding. Lactation had been stopped using cabergoline during the first days after delivery given the critical condition of the patient.

Table 1 Laboratory findings from the postpartum period and after adrenalectomy

	Postpartum (SI units)	Post surgery (SI units)
ACTH (1.584–13.99 pmol/L)	0.88	2.86
Cortisol (172.14–496.58 nmol/L)	86	69
TSH (0.27–4.2 mIU/L)	0.78	0.54
fT3 (3.30–6.32 pmol/L)	2.93	4.90
fT4 (9.78–21.36 pmol/L)	8.11	17.24
Prolactin (0.20–1.01 nmol/L)	0.56	0.41
GH (0.126–9.88 µg/L)	0.29	0.37
IGF-1 (10.63–36.43 nmol/L)	6.68	10.74
LH (2.4–12.6 IU/L)	<0.3	7.5
FSH (3.5–12.5 IU/L)	<0.3	9.2
Estradiol (98.01–572.67 pmol/L)	103	18
DHEAS (1.65–9.13 µmol/L)	0.16	0.16
Testosterone (0.27–0.166 nmol/L)	<0.1	<0.1
Metanephrine (plasma) (<329.5 pmol/L)	12 294.8	106.5
Normetanephrine (plasma) (<1070.2 pmol/L)	9920.8	305.8
Metanephrine (urine/day) (<1622.4 nmol/day)	87 041.8	136.9
Normetanephrine (urine/day) (<2129.4 nmol/day)	18 891.6	884.5

ACTH, Adrenocorticotropic Hormone ; DHEA, Dehydroepiandrosterone; FSH, Follicle-stimulating Hormone; fT3, free Triiodothyronine; fT4, free Thyroxine; GH, Growth Hormone; IGF-1, Insulin-like Growth Factor 1 ; LH, Luteinizing Hormone; TSH, Thyroid-stimulating Hormone.

In addition, the patient had developed hyponatraemia (127 mmol/L, ref: 136–145 mmol/L) and low normal serum thyrotropin while both free triiodothyronine (fT3) and free thyroxine (fT4) were reduced suggesting central hypothyroidism and potentially adrenal insufficiency (table 1). Hence, we analysed the pituitary hormones and found a complete loss of anterior pituitary function with thyrotropic, corticotropic, gonadotropic and somatotropic insufficiency but no diabetes insipidus (table 1). Thus, in addition to pheochromocytoma, the patient also presented with postpartum hypopituitarism suggesting Sheehan's syndrome. We therefore performed a pituitary MRI that showed subtotal necrosis of the anterior pituitary gland in

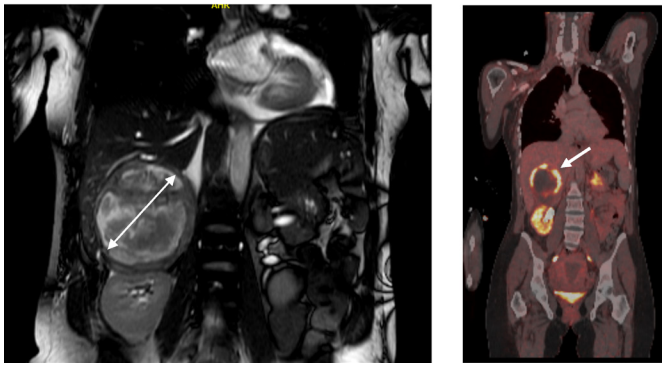

Figure 1 Both MRI (a) and F-DOPA PET/CT (b) reveal a large cystic mass in the upper right abdomen. Positive F-DOPA uptake in the PET scan (b) confirms the suspected diagnosis of haemorrhagic pheochromocytoma. F-DOPA PET/CT: 18-fluorodopa positron emission tomography CT scan.

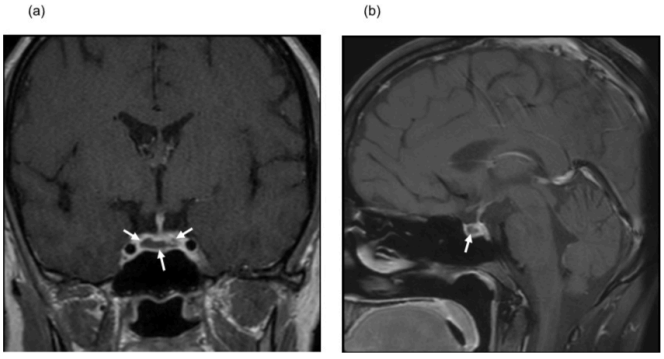

Figure 2 Pituitary MRI with paracoronal (a) and sagittal (b) T1-weighted images after contrast administration demonstrate a lack of central contrast enhancement (arrows) of the anterior pituitary lobe resulting in rim enhancement, in accordance with pituitary necrosis.

accordance with pituitary infarction and Sheehan's syndrome (figure 2). No signal changes pointing towards haemorrhage within the pituitary were found. After initiation of hydrocortisone (20 mg) and levothyroxine (50 µg) replacement, serum sodium concentrations as well as fT3 and fT4 levels normalised quickly (table 1).

The haemodynamic condition remained stable with a complete normalisation of the cardiac output, which allowed a transfer of the patient to a regular ward. Due to the recent pulmonary embolism and the necessity for anticoagulation, a surgical removal of the pheochromocytoma had to be delayed. Thus, the patient was started on an alpha-blocker (doxazosin) at the highest dose tolerated that did not cause hypotension and was discharged from the hospital. 3 months later, the pheochromocytoma was removed in toto by open abdominal surgery due to the tumour size and cystic nature (online supplemental figure 1). Histological work-up revealed a centrally necrotic and haemorrhagic pheochromocytoma of the right adrenal gland with low differentiation and high likelihood for metastasis (tumour- node-metastasis (TNM) classification: pT2, pNX, Residual tumour: R0; Pheochromocytoma of the Adrenal Gland Scaled Score (PASS) 11/20, Grading of Adrenal Pheochromocytoma and Paraganglioma (GAPP) Score 7/10 (histological pattern: large cell nest (1), cellularity: high (2), comedo necrosis: present (2), vascular/capsular invasion: present (1), Ki67 labelling index: up to 2% (1), catecholamine type: epinephrine type (epinephrine (E)+norepineprhine (NE) (0))).

DIFFERENTIAL DIAGNOSIS

The differential diagnosis of pheochromocytoma-induced cardiac failure and other forms of acute heart failure such as peripartum cardiomyopathy can propose a clinical conundrum given that symptoms and echocardiographic features can be very similar.[5] However, a history of repeated hypertensive episodes and arrhythmias, especially before pregnancy, may be suspicious for pheochromocytoma rather than peripartum cardiomyopathy.[14 15] Another possible distinction may be the initial onset of symptoms. Even though acute pheochromocytoma-associated symptoms are more common during later phases of pregnancy, they can also occur earlier during pregnancy.[16]

In contrast, one of the definitions of peripartum cardiomyopathy is the development of acute heart failure during the last month of pregnancy up to 5 months postpartum.[17]

OUTCOME AND FOLLOW-UP

The patient has been followed at the endocrine outpatient clinic for 4 years. She is in good health and working again. Genetic tests for hereditary pheochromocytoma including *SDHB, SDHC, SDHD* and *RET* as well as whole exome sequencing came back negative. Postoperative MN and NMN concentrations in plasma and urine have remained normal. F-DOPA PET/CT scans have been performed annually (according to the patient's strong wish) and have not detected any residual or recurring pheochromocytoma. Two follow-up pituitary MRIs (the latest 3.5 years after the initial diagnosis) showed an empty sella with a residual pituitary gland barely detectable on the ground and an intact pituitary stalk within the sella turcica.

The patient is still on hydrocortisone, levothyroxine as well as sex hormone replacement therapy due to her hypopituitarism. However, the levothyroxine dose could be reduced to 25 µg suggesting partial recovery of central hypothyroidism (table 1). Follow-up visits are now performed once a year.

DISCUSSION

Phaeochromocytoma crisis during pregnancy or the peripartum phase is an extremely rare but life-threatening complication, and no clinical guidelines exist to manage such a critical situation. A diagnosis of pheochromocytoma after delivery poses the highest risk for adverse maternal or fetal outcomes. Pregnant women with pheochromocytomas are therefore most vulnerable during delivery given that fetal movements, uterine contractions and labour can cause release of catecholamines with the risk for cardiovascular emergencies. Catecholamine bursts and hypertensive episodes can also disrupt placental circulation, resulting in fetal hypoxia or even miscarriage.[18] However, morbidity and mortality have improved in recent decades due to appropriate case detection and medical management.[9]

In general, the diagnosis of pheochromocytomas is often delayed due to the rarity of this condition and the sometimes non-specific clinical presentation. Particularly during pregnancy, high blood pressure, one of the leading symptoms of pheochromocytoma, could be misinterpreted as gestational hypertension. However, pheochromocytoma should be considered in women with hypertensive crisis or newly diagnosed hypertension, especially in the first trimester of pregnancy.[19 20] In our patient, hypertensive episodes before and during pregnancy had been attributed to panic attacks, which can also be a symptom of pheochromocytoma. Even if a full diagnostic work-up of various secondary forms of hypertension may not be possible during pregnancy, given that imaging techniques with radiation exposure cannot be used, the analysis of plasma fractioned (nor)metanephrines and/or 24-hour urinary fractionated (nor) metanephrines and catecholamines is feasible and should be performed if pheochromocytoma is suspected.[21 22]

In addition, case detection is recommended before pregnancy in women with a genetic predisposition for or a family history of pheochromocytoma or paraganglioma.[9] If pheochromocytoma is diagnosed during pregnancy, medical therapy is recommended to avoid hypertensive crisis. Alpha-adrenergic blockers are the treatment of choice. Antepartum surgery can be considered in the second trimester. However, a recent retrospective study showed no improved maternal or fetal outcomes, although these findings need to be interpreted with caution given the retrospective nature of the study with reported missing data, potential selection and information bias, as well as non-systematic enrolment at some centres.[18] The period of enrolment for this study spans several decades during which significant advances both in obstetric care

and understanding of pheochromocytoma occurred. In general, the study reported markedly lower adverse maternal and fetal outcomes compared with previous reports,[23 24] which further limits any conclusions about the potential risks of antepartum surgical vs non-surgical treatment.

One of the most dangerous complications of pheochromocytoma crisis is acute cardiac failure, as occurred in the case presented here. Even though the exact mechanisms are not completely understood, it has been hypothesised that elevated catecholamine levels and/or their oxidation products lead to increased permeability of the sarcolemmal membrane resulting in enhanced intracellular calcium influx and consequently myocardial cell necrosis.[25]

Another postulated mechanism implies that excess adrenergic stimulation causes vasoconstriction and decreased coronary perfusion resulting in 'ischaemic myocarditis'.[25 26]

ECMO is an effective salvage therapy for acute haemodynamic failure but is associated with high risk of bleeding, thromboembolism and neurological injury.[27 28] In this case, ECMO support was critical for the favourable outcome of the patient because unnecessary high-urgency heart transplantation could be avoided, and it allowed a thorough endocrine and imaging work-up.

During that work-up, in addition to pheochromocytoma, Sheehan's syndrome with complete anterior pituitary deficiency was discovered in our patient. The pituitary insufficiency in Sheehan's syndrome develops as a consequence of pituitary necrosis, which occurs most commonly due to haemorrhagic shock during labour, for example, after massive uterine bleeding.[29] The patient we described here had a vaginal delivery without any complications and no severe blood loss, so it is very unlikely that uterine haemorrhage was the cause of Sheehan's syndrome. However, she developed cardiogenic shock that required CPR most likely as a result of a peripartum pheochromocytoma crisis which may have led to a malperfusion of the pituitary. In addition, our patient developed several episodes of hypertension and hypotension during the first days after delivery while at the ICU and required intermittent administration of vasodilators and vasopressors. That could have further compromised the vulnerable pituitary perfusion during the postpartum phase. The increase in pituitary volume during pregnancy and the changes in the intrasellar pressure render the tissue particularly susceptible to ischaemia.[30] Hence, all facts point towards the haemodynamic consequences of pheochromocytoma crisis as the critical event for the development of Sheehan's syndrome in our patient during the most vulnerable phase after delivery. This is an unprecedented sequence of rare endocrine complications that has almost cost this young woman and her unborn child their lives. The complexity and severity of this case also highlight the importance for the management of such cases by an interdisciplinary team at a specialised centre with the appropriate experience in critical care medicine, diagnosis of rare endocrine conditions and surgical treatment of such rare tumours. For instance, the signs of Sheehan's syndrome could have been easily overlooked in this patient given that hyponatraemia and abnormalities of thyroid hormone levels are frequent in ICU patients. A surgical intervention to remove the pheochromocytoma or, let alone, high-urgency heart transplantation without appropriate perioperative hydrocortisone replacement in a patient with secondary adrenal insufficiency could have had a fatal outcome.

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Learning points

- Phaeochromocytoma crisis is a rare but life-threatening complication during pregnancy.
- Pregnant women with hypertensive crisis or newly diagnosed hypertension, especially in the first trimester of pregnancy, should be considered for screening of pheochromocytoma.
- Patients with pheochromocytoma should be managed in an experienced centre involving a multidisciplinary team.
- The signs of Sheehan's syndrome can be easily overlooked, especially when only partial pituitary insufficiency is present.

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